

This is a repository copy of *Helicobacter pylori* and cancer among adults in Uganda.

White Rose Research Online URL for this paper:

<https://eprints.whiterose.ac.uk/1872/>

---

**Article:**

Newton, R. orcid.org/0000-0001-6715-9153, Ziegler, J.L., Carpenter, L. et al. (10 more authors) (2006) *Helicobacter pylori* and cancer among adults in Uganda. *Infectious Agents and Cancers*. ISSN 1750-9378

<https://doi.org/10.1186/1750-9378-1-5>

---

**Reuse**

Items deposited in White Rose Research Online are protected by copyright, with all rights reserved unless indicated otherwise. They may be downloaded and/or printed for private study, or other acts as permitted by national copyright laws. The publisher or other rights holders may allow further reproduction and re-use of the full text version. This is indicated by the licence information on the White Rose Research Online record for the item.

**Takedown**

If you consider content in White Rose Research Online to be in breach of UK law, please notify us by emailing [eprints@whiterose.ac.uk](mailto:eprints@whiterose.ac.uk) including the URL of the record and the reason for the withdrawal request.

Short report

Open Access

## ***Helicobacter pylori* and cancer among adults in Uganda**

Robert Newton<sup>\*1,3</sup>, John L Ziegler<sup>2,8</sup>, Delphine Casabonne<sup>3</sup>,  
Lucy Carpenter<sup>4,9</sup>, Benjamin D Gold<sup>5</sup>, Marilyn Owens<sup>5</sup>, Valerie Beral<sup>3</sup>,  
Edward Mbidde<sup>2</sup>, D Maxwell Parkin<sup>6</sup>, Henry Wabinga<sup>2</sup>, Sam Mbulaiteye<sup>2</sup>,  
Harold Jaffe<sup>7</sup> and the Uganda Kaposi's Sarcoma Study Group<sup>9</sup>

Address: <sup>1</sup>Epidemiology and Genetics Unit, Dept. of Health Sciences, First Floor, Seebom Rowntree Building, University of York, Heslington, York, YO10 5DD, UK, <sup>2</sup>Uganda Cancer Institute and Makerere University Medical School, Kampala, Uganda, <sup>3</sup>Cancer Research UK Epidemiology Unit, University of Oxford, Richard Doll Building, Roosevelt Drive, Oxford, OX3 7LF, UK, <sup>4</sup>MRC Programme on AIDS, Uganda Virus Research Institute, PO Box 49, Entebbe, Uganda, <sup>5</sup>Division of Pediatric Gastroenterology and Nutrition, Department, Pediatrics, Emory University School of Medicine, 2040 Ridgewood Dr., NE, Atlanta, GA 30322, USA, <sup>6</sup>International Agency for Research on Cancer, 150 Cours Albert Thomas, Lyon, France, <sup>7</sup>Centers for Disease Control and Prevention, 1600 Clifton Road, Atlanta, Georgia, 30333, USA, <sup>8</sup>Dept. of Veterans Affairs and the University of California, San Francisco, to the International Agency for Research on Cancer, France and <sup>9</sup>Dept. of Public Health, Oxford University, to the MRC Programme on AIDS, Entebbe, Uganda

Email: Robert Newton<sup>\*</sup> - Rob.Newton@egu.york.ac.uk; John L Ziegler - ziegler@itsa.ucsf.edu;  
Delphine Casabonne - delphine.casabonne@ceu.ox.ac.uk; Lucy Carpenter - lucy.carpenter@nuffield.ox.ac.uk;  
Benjamin D Gold - ben\_gold@oz.ped.emory.edu; Marilyn Owens - ben\_gold@oz.ped.emory.edu; Valerie Beral - pa.valerie.beral@ceu.ox.ac.uk;  
Edward Mbidde - mbidde1@infocom.co.ug; D Maxwell Parkin - ctsu0138@herald.ox.ac.uk; Henry Wabinga - cancer-reg@infocom.co.uk;  
Sam Mbulaiteye - mbulaits@mail.nih.gov; Harold Jaffe - Harold.Jaffe@dphpc.ox.ac.uk; the Uganda Kaposi's Sarcoma Study Group - Rob.Newton@egu.york.ac.uk

<sup>\*</sup> Corresponding author

Published: 07 November 2006

Received: 04 May 2006

*Infectious Agents and Cancer* 2006, **1**:5 doi:10.1186/1750-9378-1-5

Accepted: 07 November 2006

This article is available from: <http://www.infectagentscancer.com/content/1/1/5>

© 2006 Newton et al; licensee BioMed Central Ltd.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/2.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

### **Abstract**

Data from Africa on infection with *Helicobacter pylori* (*H. pylori*) are sparse. Therefore, as part of an epidemiological study of cancer in Uganda, we investigated the prevalence and determinants of antibodies against *H. pylori* among 854 people with different cancer types and benign tumours. Patients were recruited from hospitals in Kampala, Uganda, interviewed about various demographic and lifestyle factors and tested for antibodies against *H. pylori*. In all patients combined, excluding those with stomach cancer (which has been associated with *H. pylori* infection), the prevalence of antibodies was 87% (723/833) overall, but declined with increasing age ( $p = 0.02$ ) and was lower among people who were HIV seropositive compared to seronegative ( $p < 0.001$ ). Otherwise, there were few consistent epidemiological associations. Among those with stomach cancer, 18/21 (86%) had anti-*H. pylori* antibodies (odds ratio 0.8, 95% confidence intervals 0.2–2.9,  $p = 0.7$ ; estimated using all other patients as controls, with adjustment for age, sex and HIV serostatus). No other cancer site or type was significantly associated with anti-*H. pylori* antibodies. The prevalence of *H. pylori* reported here is broadly in accord with results from other developing countries, although the determinants of infection and its' role in the aetiology of gastric cancer in Uganda remain unclear.

### **Background**

The work described in this report was part of an epidemi-

ological study of cancer in Kampala, Uganda [1-7]. Data from Africa on infection with *Helicobacter pylori* (*H. pylori*)

are sparse. Therefore, we examine here the role of antibodies against *H. pylori* in relation to the risk of cancer and investigate the prevalence and determinants of infection among 854 people with different cancer types and benign tumours.

## Materials and methods

Full details of the methods are provided elsewhere [1,2]. Briefly between 1994 and 1998, we recruited adults 15 years or older with a new diagnosis of cancer from the wards and out-patient clinics of the main hospitals in Kampala, Uganda. After informed consent and counselling, patients were interviewed and tested for infection with HIV-1 using the Cambridge Bioscience Recombigen ELISA (Cambridge, MA) on sera or the GACELISA method (Murex, Dartford, UK) on saliva. Cancer diagnoses were established by histology or other laboratory investigation, where possible. Diagnoses made on clinical grounds alone were reviewed by the investigators. The study was approved by the Committee on Human Research (VA Medical Centre and University of California San Francisco) and by the Uganda National Council for Science and Technology.

Following HIV testing, remaining sera were stored at minus 80 Celsius and were later shipped on dry ice to the Centres for Disease Control and Prevention, Atlanta, USA, for *H. pylori* testing. Assays were performed by a single investigator who was blind to the diagnosis of the patient from whom the blood was obtained. Briefly, *H. pylori* organisms were grown overnight in brucella broth (GIBCO Laboratories, Madison, WI) with 10% fetal bovine serum (Sigma, St. Louis, MO), 5 µg/ml trimethoprim and 10 µg/ml vancomycin (Sigma). *H. pylori* antigen extraction and protein isolation were done by gentle freeze-thaw sonication (Heat System, Farmingdale, NY) [8,9]. A standard protein assay (Pierce, Rockford, IL) was used to determine the accurate and reproducible quantity of solid-phase antigen for our microtitre research ELISA [10]. Cross-reactivity and specificity of *H. pylori* whole-cell antigens has been described previously [9,10]. Optical density (OD) values at a wavelength of 492 nm were determined in triplicate for each biopsy-confirmed control patient sera, using a standard 96-well microtiter plate ELISA spectrophotometer (Fisher Scientific, Pittsburgh, PA). The mean OD values were then calculated. The ELISA cut-off values were derived using known *H. pylori*-positive and negative control sera as previously described [9,11]. In previous validation studies the assay has demonstrated a high and reproducible sensitivity and specificity in African patients as compared to upper endoscopy and biopsy; sensitivity >88%, specificity >90% [9,11].

Serological results were available for 50 people with non-malignant manifestations of HIV disease, recruited from

the out-patient department of Mulago hospital and for 804 patients with cancer or benign tumours, for whom a stored blood sample was available for testing. The latter group comprised people with cancers of the oral cavity (26), oesophagus (38), stomach (21), liver (52), skin (22), breast (69), cervix (190), ovary (22), prostate (10), penis (14), eye (63), and non-Hodgkin's lymphoma (46), Hodgkin's disease (24), Kaposi's sarcoma (46), other cancer sites or types (126) and benign tumours (35).

Data were computerised by trained clerks using EPI-INFO software (CDC, Atlanta) and statistical analyses were conducted using STATA (STATA Corporation, Texas). Only a small proportion of those tested were seronegative for antibodies against *H. pylori* (optical density <0.9) or had an indeterminate result (optical density 0.9–1.3). In all analyses, those with indeterminate results were considered to be seronegative. In order to examine potential confounding factors, the risk of being seropositive for antibodies against *H. pylori* was examined in relation to various social and demographic factors among all patients combined (but excluding stomach cancer, which has been associated with *H. pylori* infection). Odds ratios (OR) were estimated using unconditional logistic regression modelling with adjustment for sex, age group (<30, 30–45, 46+) and HIV serostatus. When calculating odds ratios in relation to anti-*H. pylori* antibodies, for each cancer site or type, the comparison group included all other patients with the exception of stomach cancer. Tests for association used the  $\chi^2$  test for linear trend on one degree of freedom and all p values are 2-sided. Risk factors for high titres of antibodies against *H. pylori* were examined amongst all patients combined (excluding stomach cancer), but no clear associations were identified and the data are not shown.

## Results

*H. pylori* antibody status was available for 854 people; 87% (741) were seropositive, 4% (38) were seronegative and 9% (75) had an indeterminate result. Table 1 shows the association between *H. pylori* serostatus and selected social and demographic factors among all patients excluding those with stomach cancer. The prevalence of antibodies did not vary with sex, but declined with increasing age ( $\chi^2_1 = 5.1$ ,  $p = 0.02$ ) and was lower among people who were HIV seropositive compared to seronegative ( $\chi^2_1 = 16.2$ ,  $p < 0.001$ ). No other factor examined was associated with antibodies against *H. pylori* with the exception of religion: the prevalence of antibodies was higher among Muslims than among Christians ( $\chi^2_1 = 5.6$ ,  $p = 0.02$ ).

Table 2 shows the association between anti-*H. pylori* antibodies and specific cancer sites or types, together with the proportion of cancers with a laboratory verification of diagnosis. Overall, 62% of cancers were diagnosed on the

**Table 1: Odds ratios (OR) for *H. pylori* seropositivity according to various social and demographic factors among all patients (excluding those with stomach cancer)**

Variable	Number positive/ total	OR (95% CI) <sup>1</sup>	Variable	Number positive/ total	OR (95% CI) <sup>1</sup>
<b>Sex</b>			<b>Time to market</b>		
Male	247/284	1.0	<30 minutes	323/375	1.0
Female	476/549	0.9 (0.6–1.3)	30+ minutes	278/320	1.0 (0.7–1.6)
		$\chi^2_1 = 0.5, p = 0.5$			$\chi^2_1 = 0.0, p = 0.9$
<b>Age group</b>			<b>Size of community</b>		
<36 years	269/309	1.0	>100 houses	220/262	1.0
36–50 years	250/282	1.0 (0.6–1.6)	10–99 houses	421/482	1.3 (0.9–2.1)
51+ years	204/242	0.5 (0.3–0.9)	<10 houses	51/57	1.6 (0.6–4.1)
		$\chi^2_1 = 5.1, p = 0.02$			$\chi^2_1 = 1.0, p = 0.3$
<b>HIV serostatus</b>			<b>Ever travel from home</b>		
Negative	514/574	1.0	Yes	91/109	1.0
Positive	201/250	0.4 (0.3–0.6)	No	599/688	1.4 (0.7–2.5)
		$\chi^2_1 = 16.2, p < 0.001$			$\chi^2_1 = 1.0, p = 0.3$
<b>Region of residence</b>			<b>Household size</b>		
Kampala	161/188	1.0	<6 people	357/410	1.0
Rest of Uganda	560/643	1.0 (0.6–1.6)	6+ people	354/411	0.8 (0.5–1.2)
		$\chi^2_1 = 0.0, p = 0.9$			$\chi^2_1 = 1.6, p = 0.2$
<b>Tribe</b>			<b>Number of siblings</b>		
Baganda	349/410	1.0	<6 siblings	379/438	1.0
Other	374/423	1.2 (0.8–1.8)	6+ siblings	333/384	0.9 (0.6–1.4)
		$\chi^2_1 = 0.8, p = 0.4$			$\chi^2_1 = 0.1, p = 0.8$
<b>Religion</b>			<b>Number of children</b>		
Muslim	95/100	1.0	<7 children	341/397	1.0
Christian	619/723	0.3 (0.1–0.8)	7+ children	290/329	1.1 (0.7–1.8)
		$\chi^2_1 = 5.6, p = 0.02$			$\chi^2_1 = 0.1, p = 0.8$
<b>Occupation</b>			<b>Tobacco consumption</b>		
Cultivator	334/387	1.0	Never smoker	562/647	1.0
Other	382/439	1.1 (0.7–1.9)	Past smoker	81/92	1.2 (0.6–2.5)
		$\chi^2_1 = 0.1, p = 0.8$	Current smoker	68/82	0.7 (0.4–1.4)
					$\chi^2_1 = 0.5, p = 0.5$
<b>Education level</b>			<b>Alcohol consumption</b>		
No school	153/175	1.0	Never	391/443	1.0
Primary	361/415	0.9 (0.5–1.6)	About once/week	134/157	0.8 (0.5–1.4)
Secondary/tertiary	191/225	0.7 (0.4–1.4)	2–4 days/week	91/106	0.9 (0.5–1.6)
		$\chi^2_1 = 1.0, p = 0.3$	Most days	93/113	0.7 (0.4–1.2)
					$\chi^2_1 = 1.6, p = 0.2$
<b>Household Income (Ug. Sh.)</b>			<b>Lifetime number of sexual partners</b>		
15,000+	464/533	1.0	1–2 partners	182/204	1.0
<15,000	196/230	0.9 (0.6–1.5)	3–9 partners	305/352	0.9 (0.5–1.5)
		$\chi^2_1 = 0.1, p = 0.8$	10+ partners	196/232	0.7 (0.4–1.3)
					$\chi^2_1 = 1.2, p = 0.3$

1. Odds ratios adjusted for age group (<30, 30–45, 46+), sex and HIV serostatus

basis of histology or other laboratory investigation, but the figure varied by cancer site or type, being lowest for prostate cancer (44%) and highest for Kaposi's sarcoma (91%). Of those people with stomach cancer, 90% (19/

21) had the diagnosis confirmed by a laboratory investigation.

Among 21 cases with stomach cancer, one was HIV seropositive, 13 were women, one was aged <30 years, three

**Table 2: *H. pylori* serostatus for different cancer sites or types and non-malignant conditions, together with the percentage of each cancer with laboratory verification of diagnosis**

Cancer site or type	Percentage with laboratory verification of diagnosis	Number anti- <i>H. pylori</i> anti-body positive/total	Odds ratio (95% CI) <sup>1</sup>	$\chi^2_1$ and p value
<b>Stomach</b>	90%	18/21	0.8 (0.2–2.9)	$\chi^2_1 = 0.1$ , p = 0.7
<b>All controls<sup>2</sup></b>	-	723/833	1.0	-
<b>Oral</b>	56%	21/26	0.6 (0.2–1.7)	$\chi^2_1 = 0.9$ , p = 0.4
<b>Oesophagus</b>	45%	37/38	5.1 (0.7–38)	$\chi^2_1 = 2.5$ , p = 0.1
<b>Liver</b>	56%	44/52	0.7 (0.3–1.6)	$\chi^2_1 = 0.8$ , p = 0.4
<b>Skin</b>	77%	18/22	0.6 (0.2–1.9)	$\chi^2_1 = 0.8$ , p = 0.4
<b>Breast</b>	62%	62/69	1.4 (0.6–3.5)	$\chi^2_1 = 0.6$ , p = 0.4
<b>Cervix</b>	50%	173/190	1.6 (0.9–2.8)	$\chi^2_1 = 2.1$ , p = 0.2
<b>Ovary</b>	73%	20/22	1.1 (0.3–5.0)	$\chi^2_1 = 0.0$ , p = 0.9
<b>Prostate</b>	44%	7/10	0.3 (0.1–1.4)	$\chi^2_1 = 2.4$ , p = 0.1
<b>Penis</b>	57%	12/14	0.9 (0.2–4.3)	$\chi^2_1 = 0.0$ , p = 0.9
<b>Conjunctiva</b>	66%	33/38	1.3 (0.5–3.5)	$\chi^2_1 = 0.3$ , p = 0.6
<b>Other eye</b>	52%	22/25	1.1 (0.3–3.9)	$\chi^2_1 = 0.0$ , p = 0.9
<b>Non-Hodgkin's lymphoma</b>	76%	39/46	0.8 (0.3–1.8)	$\chi^2_1 = 0.4$ , p = 0.5
<b>Hodgkin's lymphoma</b>	83%	20/24	0.6 (0.2–1.9)	$\chi^2_1 = 0.7$ , p = 0.4
<b>Kaposi's sarcoma</b>	91%	36/46	0.7 (0.3–1.6)	$\chi^2_1 = 0.9$ , p = 0.4

1. Odds ratios adjusted for age group (<30, 30–45, 46+), sex and HIV serostatus

2. The comparison group for the calculation of odds ratios includes all other cancers and non-malignant conditions, excluding stomach cancer

Note: *H. Pylori* serostatus: negative – optical density (O.D.) 0.0–0.8; indeterminate – O.D. 0.9–1.3; positive – O.D. 1.4–4.0. For the purposes of this analysis, those with an indeterminate result were considered to be seronegative.

were aged between 30–45 years and 17 were aged 46+ years. Among those with stomach cancer, 86% (18/21) were seropositive for antibodies against *H. pylori* antigens, compared to 87% (761/871) of the comparison group (odds ratio = 0.8, 95% confidence intervals 0.2–2.9;  $\chi^2_1 = 0.1$ , p = 0.7). Nor was there a statistically significant association between anti-*H. pylori* antibodies and any other cancer site or type examined.

## Discussion

Here we report the first data from Uganda on the seroprevalence of antibodies against *H. pylori*. The prevalence of 87% was broadly comparable to that reported from other hospital series elsewhere on the African continent: 79% in Algeria [12], 71% in Côte d'Ivoire [12], 79% in the Democratic Republic of Congo [13], 85% in Nigeria [14] and 86–93% in South African blacks [15,16]. In the only other study of *H. pylori* from Uganda, Wabinga [17] identified a high frequency of colonisation in gastric endoscopic biopsies from people with upper gastrointestinal symptoms.

Data on the determinants of infection with *H. pylori* in Africa are scant. The prevalence of infection has been found to rise through childhood, reaching over 70% in early adulthood [15] and has been associated with markers of Hepatitis A infection [15], premastication of infant's food [18] and low social class in some studies, but not others (reviewed in reference [19]). In this study, few consistent associations between either the prevalence or titre of anti-*H. pylori* antibodies and any of the risk factors

examined were identified. The reasons for the lower prevalence identified in this study among people aged over 50 years and among HIV infected people are unclear. Similarly, since no consistent differences between religious groups have been identified to date in this study [1–7], it is likely that the differences in the prevalence of anti-*H. pylori* antibodies observed here, between Christians and Muslims, arose by chance.

It is recognised that serum antibodies against *H. pylori* may decline because of the development of gastric changes, such as malignancy, that can suppress or kill the infection [20]. Case-control studies have therefore shown inconsistent associations between antibodies against *H. pylori* and gastric cancer (reviewed in reference [21]). The lack of an association in this study and in the only other from Africa in which serum antibodies were measured [22] is, therefore, unsurprising. The lack of statistical power in this study (based on only 21 cases of stomach cancer) and the incomplete diagnostic verification may have further reduced the ability to detect an association. Indeed, the only study from Africa to find an association between gastric cancer and *H. pylori* involved assessment of infection status microscopically in tissue taken from areas adjacent to disease and included only six people with the tumour [23].

The apparent increase in the incidence of gastric cancer seen in Uganda since the 1960s [24], though not statistically significant, may have been influenced by improve-

ments in diagnosis and is at odds with the decline seen throughout much of the rest of the world. The role of *H. pylori* and other factors in the aetiology of gastric cancer in Uganda and elsewhere in Africa remains unclear.

## Acknowledgements

Support for this work was provided by Mulago Hospital and Makerere Medical School, Kampala, Uganda, the Imperial Cancer Research Fund (now Cancer Research UK), the United States Centers for Disease Control and Prevention (interagency agreement with the Department of Veterans Affairs) and the International Agency for Research on Cancer, World Health Organisation, Lyon.

The Uganda Kaposi's Sarcoma Study Group includes named authors and V Sembajwe, M Kalinaki, R Byansi, C Rwatooro, S Namboozee, B Tushemeirwe, N Byabazaire (deceased), E Bitamazire, E Katabira, J Mugerwa (deceased), D Tindyebwa, C Ateenyi-Agaba, L Marum, J Whitworth, B Richardson, G Reeves, R Weiss and K de Cock.

## References

1. Newton R, Ziegler J, Beral V, Mbidde E, Carpenter L, Wabinga H, Mbulaitaye S, Appleby P, Reeves G, Jaffe H, the Uganda Kaposi's Sarcoma Study Group: **A case-control study of Human Immunodeficiency Virus infection and cancer in adults and children residing in Kampala, Uganda.** *Int J Cancer* 2001, **92**:622-627.
2. Newton R, Ziegler J, Ateenyi-Agaba C, Bousarghin L, Casabonne D, Beral V, Mbidde E, Carpenter L, Reeves G, Parkin DM, Wabinga H, Mbulaitaye S, Jaffe H, Bourboullia D, Boshoff C, Coursaget P, the Uganda Kaposi's Sarcoma Study Group: **The epidemiology of conjunctival squamous cell carcinoma in Uganda.** *Br J Cancer* 2002, **87**:301-308.
3. Newton R, Ziegler J, Bourboullia D, Casabonne D, Beral V, Mbidde E, Carpenter L, Reeves G, Parkin DM, Wabinga H, Mbulaitaye S, Jaffe H, the Uganda Kaposi's Sarcoma Study Group, Weiss R, Boshoff C: **The sero-epidemiology of Kaposi's sarcoma-associated herpesvirus in adults with cancer, in Uganda.** *Int J Cancer* 2003, **103**:226-232.
4. Newton R, Ziegler J, Bourboullia D, Casabonne D, Beral V, Mbidde E, Carpenter L, Parkin DM, Wabinga H, Mbulaitaye S, Jaffe H, the Uganda Kaposi's Sarcoma Study Group, Weiss R, Boshoff C: **Infection with Kaposi's sarcoma-associated herpesvirus (KSHV) and human immunodeficiency virus (HIV) in relation to the risk and clinical presentation of Kaposi's sarcoma in Uganda.** *Br J Cancer* 2003, **89**(3):502-504.
5. Newton R, Bousarghin L, Ziegler J, Casabonne D, Beral V, Mbidde E, Carpenter L, Parkin DM, Wabinga H, Mbulaitaye S, Jaffe H, Touzé A, Coursaget P, the Uganda Kaposi's Sarcoma Study Group: **Human papillomaviruses and cancer in Uganda.** *Eur J Cancer Prev* 2004, **13**(2):113-118.
6. Ziegler JL, Newton R, Katongole-Mbidde E, Mbulaitaye S, DeCock K, Wabinga H, Mugerwa J, Katabira E, Jaffe H, Parkin DM, Reeves G, Beral V, for the Uganda Kaposi's sarcoma study group: **Risk factors for HIV-associated Kaposi's sarcoma in Uganda: a case-control study of 1026 Adults.** *AIDS* 1997, **11**:1619-1626.
7. Ziegler J, Newton R, Bourboullia D, Casabonne D, Beral V, Mbidde E, Carpenter L, Reeves G, Wabinga H, Mbulaitaye S, Jaffe H, the Uganda Kaposi's Sarcoma Study Group, Weiss R, Boshoff C: **Risk factors for Kaposi's sarcoma: a case-control study of HIV seronegative people in Uganda.** *Int J Cancer* 2003, **103**:233-240.
8. Pei Z, Ellison RT, Blaser MJ: **Identification, purification and characterization of major antigenic proteins of Campylobacter jejuni.** *J Biol Chem* 1991, **266**:1663-1669.
9. Khanna B, Cutler A, Israel NR, Perry M, Lastovica A, Fields PI, Gold BD: **Use caution with serologic testing for Helicobacter pylori infection in children.** *J Infect Dis* 1988, **178**:460-465.
10. Perez-Perez GI, Dwoskin BM, Chadon JE, Blaser MJ: **Campylobacter pylori antibodies in humans.** *Ann Intern Med* 1998, **109**:11-17.
11. Gold BD, Khanna B, Huang LM, Lee C-Y, Banatvala N: **Helicobacter pylori acquisition in infancy after decline of maternal passive immunity.** *Pediatric Research* 1997, **41**:641-646.
12. Megraud F, Brassens-Rabbe MP, Denis F, Belbourni A, Hoa DQ: **Seroepidemiology of Campylobacter pylori infection in various populations.** *J Clin Microbiol* 1989, **27**:1870-73.
13. Glupczynski Y, Bourdeaux L, Verhas M, DePrez C, DeVos D, Devreker T: **Use of a urea breath test versus invasive methods to determine the prevalence of Helicobacter pylori in Zaire.** *Eur J Clin Microbiol Infect Dis* 1992, **11**:322-27.
14. Holcombe C, Omotara BA, Eldridge J, Jones DM: **H. pylori, the most common bacterial infection in Africa: a random serological study.** *Am J Gastroenterol* 1992, **87**:28-30.
15. Sathar MA, Simjee AE, Wittenberg DF, Mayat AM: **Seroprevalence of Helicobacter pylori infection in Natal/KwaZulu, South Africa.** *Eur J Gastroenterol Hepatol* 1994, **6**:37-41.
16. Sitas F, Sathar MA, Simjee AE, Lombard CJ, Steyn K, Badenhorst CJ, Jooste PL, Bourne L: **Helicobacter pylori seroprevalence in the African adult population of the Cape Peninsula.** *S Afr J Epidemiol Infect* 1997, **12**:111-114.
17. Wabinga HR: **Frequency of Helicobacter pylori in gastric biopsy of Ugandan African.** *E Afr Med* 1996, **71**:691-3.
18. Albengue M, Tall F, Dabis F: **Epidemiological study of Helicobacter pylori transmission from mother to child in Africa.** *Abstract. Rev Esp Enferm Apar Dig* 1990, **78**:48.
19. Parkin DM, Ferlay J, Hamdi-Chérif, Sitas F, Thomas JO, Wabinga H, Whelan SL: **Cancer in Africa: epidemiology and prevention, IARC Scientific Publications No. 153** IARC Press, Lyon; 2003:371-6.
20. Forman D: **The prevalence of Helicobacter pylori infection in gastric cancer.** *Alimentary Pharmacology and Therapeutics* 1995, **9**(2):71-6.
21. Danesh J: **Is Helicobacter pylori infection a cause of gastric neoplasia.** In *Cancer Surveys, Infections and Human cancer Volume 33*. Edited by: Newton R, Beral V, Weiss R. Cold Spring Harbor Laboratory Press; 1999.
22. Louw JA, Kidd MSG, Kummer AF, Taylor K, Kotze U, Hanslo D: **The relationship between Helicobacter pylori infection, the virulence genotypes of the infecting strain and gastric cancer in the African setting.** *Helicobacter* 2001, **6**:268-273.
23. Jaskiewicz K, Lowrens HD, Woodroof CW, van Wyk MJ, Price SK: **The association of Campylobacter pylori with mucosal pathological changes in a population at risk of gastric cancer.** *S Afr Med J* 1989, **75**:417-419.
24. Wabinga HR, Parkin DM, Wabwire-Mangen F, Namboozee S: **Trends in cancer incidence in Kyadondo County, Uganda, 1960-1997.** *Br J Cancer* 2000, **82**:172.

Publish with **BioMed Central** and every scientist can read your work free of charge

"BioMed Central will be the most significant development for disseminating the results of biomedical research in our lifetime."

Sir Paul Nurse, Cancer Research UK

Your research papers will be:

- available free of charge to the entire biomedical community
- peer reviewed and published immediately upon acceptance
- cited in PubMed and archived on PubMed Central
- yours — you keep the copyright

Submit your manuscript here:  
http://www.biomedcentral.com/info/publishing\_adv.asp

